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Peer-Reviewed Case Report

Paranormal activity: Para-Esophageal/Gastric Hemorrhage in a Patient with a HeartMate II LVAD

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Abstract

In this report we present an unusual case of non-traumatic, spontaneous para-esophageal/para-gastric hemorrhage requiring multiple units of red blood cells in a female patient with a continuous flow left ventricular assist device. She presented with dysphagia and atypical chest pain 18 months post implantation, on the recommended anticoagulation regimen for HeartMate II support of ASA 81mg and international normalized ratio goal (INR) of 2-3 and was successfully managed with conservation support, volume resuscitation and multiple transfusions. Non-traumatic causes of acute bleeding, including acquired platelet dysfunction are considered.

Keywords: left ventricular assist device, bleeding, complications, heart failure

Introduction

Mechanical circulatory support in the form of left ventricular assist devices (LVADs) have become the standard-of-care for selected patients with end-stage heart failure refractory to medical therapy. Despite the improved survival in patients with the evolution in design from pulsatile flow to continuous flow (CF) devices, bleeding complications remain a significant problem, not the least of which is gastrointestinal (GI) bleeding.¹ In addition to the risks of bleeding related to anticoagulation, GI bleeding in CF LVADs has been attributed to the development of acquired von Willebrand disease (aVWD), diminished pulse pressure and pulsatility, advanced age, history of peptic ulcer disease, and



increased pump speed.²⁻⁴ Evidence of spontaneous bleeding from other anatomic sites are less well-described. We present a case of a spontaneous para-esophageal/gastric hemorrhage in a patient with a CF LVAD who was greater than one year post device implantation.

Case report

The patient was a 38 year old African American female with a history of non-ischemic cardiomyopathy status post HeartMate II in February of 2015, obesity (BMI of 48), and dysfunctional uterine bleeding after LVAD controlled with an intrauterine device. The patient reported her usual state of health and had no significant prior LVAD complications until the day of presentation when she awoke with sudden onset, sharp, severe chest and back pain, dysphagia, nausea, and vomiting with clear emesis. Computed tomography with angiogram (CTA) of the chest revealed no signs of dissection, but did reveal a 7.5 x5.9 cm hypodense lesion posterior to the left lobe of the liver and anterior to the stomach, with mild dilation of the distal esophagus and air/fluid levels seen in the proximal esophagus. Initial labs were notable for an INR of 2.86 and hemoglobin of 8.4g/dl (baseline ~9.5g/dl), which rapidly decreased to 6.7g/dl over the next few hours. The severe chest pain and vomiting continued, and with the assistance of the gastrointestinal (GI) team, a nasogastric tube was endoscopically placed, and visual examination revealed severe, diffuse mucosal changes concerning for ischemia from external compression. A subsequent barium swallow revealed a large mass concerning for hematoma, likely external to the esophagus, causing severe luminal narrowing and a high degree of obstruction (Figure 1). Repeat CTA revealed a much larger (9.1x 4.5x 10cm), evolving hematoma (Figure 2) which also resulted in gastric compression (Figure 3).



Figure 1. Esophagram revealing para-esophageal hematoma with esophageal obstruction

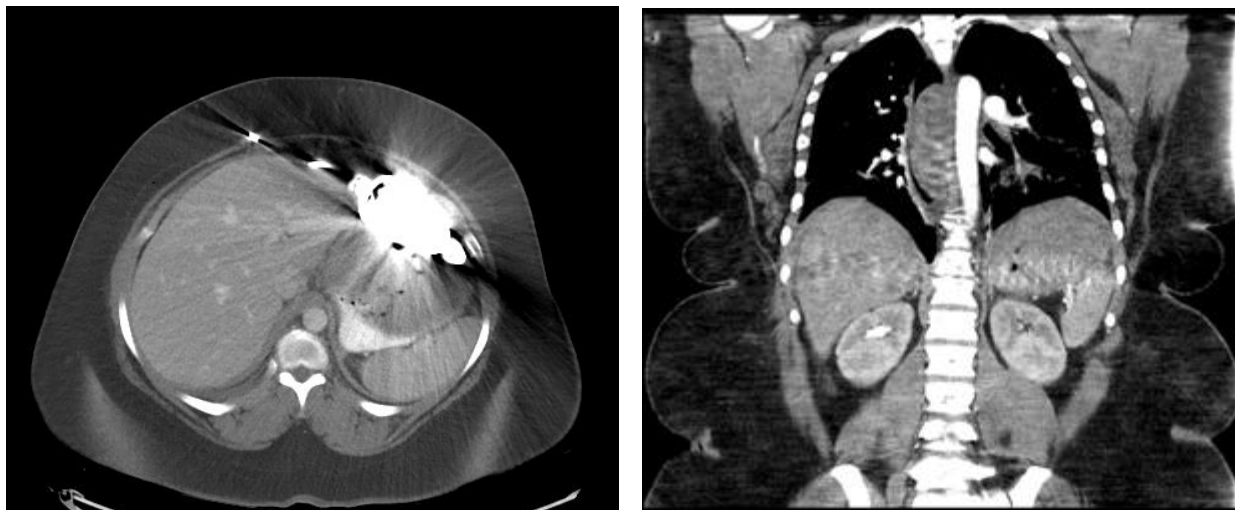


Figure 2. Cross and coronal sections of CTA of chest revealing paraesophageal and para-gastric hematoma with LVAD.



Figure 3. Sagittal section of CT with oral contrast revealing paraesophageal/paragastric hematoma causing gastric compression.



After volume and transfusion resuscitation, reversal of anticoagulation with fresh frozen plasma, and surgical evaluation, conservative management was undertaken given her clinical stability with no other source of bleeding despite interventional angiographic evaluation. She then tolerated increasing doses of intravenous heparin, was bridged to warfarin and kept nothing per os (NPO) other than medications for several days due to concerns over ischemic esophagitis. She slowly progressed to mechanical soft diet at time of discharge with mild residual dysphagia, 16 days after presentation.

Discussion

The association between CF LVADs and bleeding diatheses are well-known, in part due to the aforementioned phenomenon of aVWD, resulting from the sheer stress on the von Willebrand factor from the CF-LVAD leading to platelet dysfunction.⁵ Another mechanism independent and specifically related to the increased risk of GI bleeding in CF LVADs is similar to that of Heyde's syndrome seen in aortic stenosis. Heyde's syndrome is the theory of an abnormal pulse wave resulting in the distention of sub-mucosal venous plexuses of the GI tract, subsequent angiodysplasia and arteriovenous malformations (AVMs) with poorly perfused, and friable gastrointestinal mucosa.⁶ To our knowledge, this is the first report of a spontaneous paraesophageal/mucosal bleed that is not related to the common GI or perioperative bleeding reported in this population. Understanding the other potential mechanisms and risk factors for increased bleeding in LVAD patients is crucial to the prevention and treatment of morbidity and mortality in this already high risk population.

How sex or BMI influences the risks of these types of bleeds have yet to be elucidated. Recently, an increased incidence of thrombotic and hemorrhagic events have been identified in the female HeartMate II population as compared to males,⁷ with a more recent single center report identifying an increase in bleeding complications in females regardless of CF-LVAD type.⁸ Additionally, there is a higher risk of bleeding in patients with lower body mass index (BMI), which is clearly not the case for this patient with a BMI of 48.⁹ Taking all of this into consideration along with an INR within therapeutic range, determining the etiology of our patient's spontaneous paraesophageal/mucosal bleed remains a challenge.

Currently the mainstay of prevention for bleeding in LVAD patients is mechanical and pharmacologic intervention including monitoring INRs closely and reducing pump speed, which may be associated with decreased bleeding.⁴ Blood pressure control is important in reducing risk of intracerebral bleeding. Determining the risks, prevention and treatment of bleeding not only impacts short-term morbidity, but also has several long-term implications. For example, patients with LVADs who have experienced GI bleeding have a decrease in future heart transplant probability. While the mechanism of this relationship is unclear, speculation regarding allosensitization from receiving blood products prior to heart transplant is purported.¹⁰ Further research and reports on bleeding in this patient population is crucial to discovering targeted methods for the prevention and treatment of bleeding and its associated implications on morbidity and mortality in the LVAD population.



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